

QTc interval and electrocardiographic findings of COVID-19 patients

Electrocardiographic findings of COVID-19 patients

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Aim: This study aimed to investigate the effectiveness of using the QTc interval and electrocardiographic (ECG) findings to predict 28-day all-cause mortality in patients with COVID-19.

Material and Methods: Patients aged 18 or older who visited ED with complaints of fever, cough and shortness of breath were tested using real-time reverse-transcriptase polymerase chain reaction, were imaged with CCT, underwent ECG, and consequently, diagnosed with COVID-19 were included in this study.

Results: A total of 276 patients were included in the study. When at least one comorbid disease, reduced oxygen saturation, ECG findings of prolonged QTc interval, ventricular tachycardia/fibrillation, left bundle branch block and ST segment elevation/depression or severe lung involvement (four or five lobes) on CCT scans were detected, patients had a higher 28-day all-cause mortality rate. Compared to surviving individuals, deceased patients had approximately 4.5-fold increased D-dimer levels, and approximately 5-fold increased C-reactive protein and troponin T levels. Among the deceased patients, 40% had sinus tachycardia.

Discussion: Usage of comorbidities, ECG, laboratory tests and CCT together is useful for predicting 28-day all-cause mortality rate in patients diagnosed with COVID-19.

Keywords

COVID-19; Electrocardiography; Mortality; Chest computed tomography; Laboratory tests

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Introduction

In December 2019, a pandemic named COVID-19 pneumonia, which is caused by the novel coronavirus (SARS-nCoV-2), emerged in the city of Hubei, Wuhan, China [1]. Among the first published cohort from the Wuhan Jinyintan Hospital, which consisted of 41 patients with COVID-19 pneumonia, six (14.6%) patients' medical conditions deteriorated rapidly, leading to death from multiple organ failure [2]. When the cohort size reached 99 cases, 11 (11.1%) patients died [3]. In another Wuhan cohort comprised of patients hospitalized due to COVID-19 pneumonia, general mortality rate was reported as 4.3% (6/138) [4]. These studies have suggested that advanced age and underlying comorbidities are associated with disease severity and mortality rate in patients with COVID-19 pneumonia [2-4].

COVID-19 continues to spread rapidly throughout the world. The number of patients is growing each day, and these patients constitute the majority of cases who visit emergency departments (ED). This exceeding volume of patients hinders routine operations at ED. Establishing markers for the early and accurate diagnosis of COVID-19 and anticipating patients at risk of mortality would reduce rates of morbidity and mortality. This study aimed to investigate the effectiveness of using the QTc interval and electrocardiographic (ECG) findings to predict 28-day all-cause mortality in patients with COVID-19.

Material and Methods

Ethical considerations

This study was initiated following approval from the COVID-19 Scientific Research Review Commission (under the General Directorate of Health Services, the Ministry of Health) and the Hospital Ethical Committee (2011-KAEK-25 2020/07-18).

Study population

Patients aged 18 or older who visited ED between 15.03.2020 and 30.06.2020 with complaints of fever, cough and shortness of breath, underwent ECG, were tested with real-time reverse-transcriptase polymerase chain reaction (rRT-PCR), were imaged with chest computed tomography (CCT), and consequently, diagnosed with COVID-19 were included in the study.

Study Data

Patient information was gathered from the hospital automation

system and patient files. The assessment of CCT scans of the participating patients was based on official reports on the hospital automation system made by Radiology Specialist. The assessment of ECGs was made by two experienced Emergency Medicine Specialists. The exclusion criteria were as follows: absence of ECG, rRT-PCR or CCT results, age below 18, transfer of the patient from ED or from hospitalized clinic to another facility, and lacking patient information.

A standard data form was prepared for the study. File number, age, gender, initial complaints, existing disorders (hypertension, diabetes mellitus, coronary artery disease, chronic obstructive pulmonary disease/asthma, chronic renal failure, history of cerebrovascular accident, malignancy), ECG findings (QTc interval, heart rate, heart rhythm, bundle branch block, axis deviation, negative T wave, ST elevation or depression, pathological Q wave), laboratory findings, CCT assessment (pneumonic infiltration, localization, number of involved lobe), clinical outcome (admission to a hospital ward, admission to the intensive care unit, death) and the 28-day all-cause mortality condition of the patients were recorded in these forms.

Statistical Analysis

The data obtained were analyzed using the IBM SPSS Statistics version 21.0 software package (SPSS Inc., Chicago, IL, USA). The normality of the distribution of numeric data was evaluated with the Kolmogorov-Smirnov test. Numeric data without normal distribution, a comparison of which was made using the Mann-Whitney U test, were shown as the median and interquartile range (IQR 25-75). Normally distributed data were given as mean and standard deviation and were assessed with the independent samples Student's t-test. Categorical variables were shown as number and percentage (%). Relations between categorical variables were analyzed using the chi-squared test and Fisher's exact test. A p- value of <0.05 was considered statistically significant.

Results

A total of 276 patients were included in the study. One hundred fifty (54.3%) patients were male and 126 (45.7%) were female. The mean age of the study group was 68 (60-79) years. The most common initial complaints were shortness of breath (n=145; 52.5%), cough (n=124; 44.9%), fever (n=107; 38.8%)

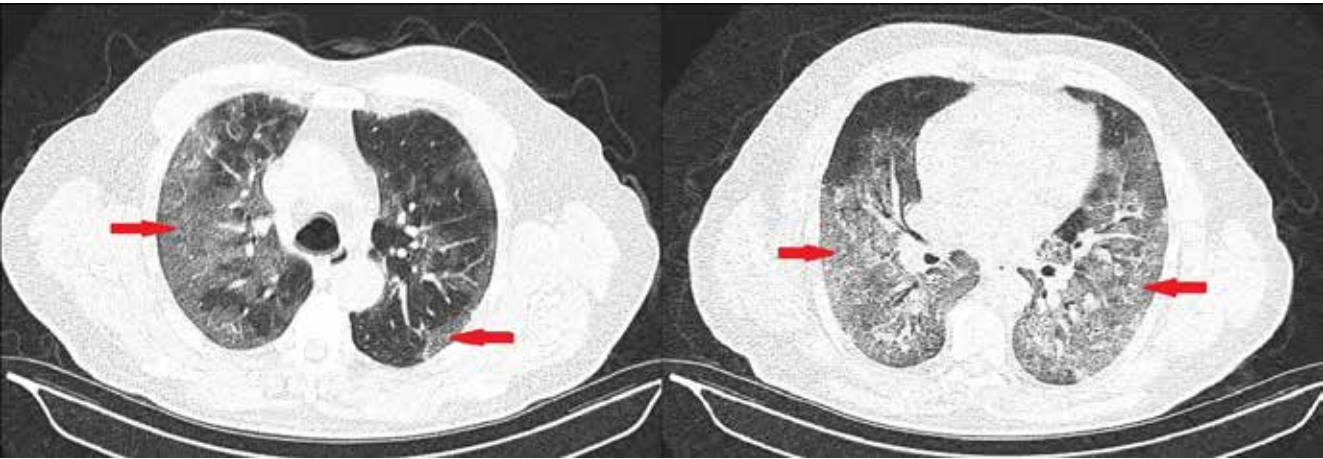


Figure 1. Thorax CT images typical of COVID-19 pneumonia in a 58-year-old man presenting with coughing, shortness of breath, and O2 saturation of 89% on room air requiring supplemental oxygen. The patient had no comorbid diseases. COVID-19 pneumonia was confirmed with rRT-PCR. Bilateral diffuse ground glass opacities (red arrows) are seen on images.

Table 1. Patients’ demographic data, symptoms, comorbidities and vital signs at admission

Total	28-day all-cause mortality			p-value
	All patients n (%) 276 (100)	Survivor n (%) 246 (89.1)	Deceased n (%) 30 (10.9)	
Age (years), median (IQR)	68 (60-79)	68 (60-78)	73 (64-81)	0.087*
Gender, n%				
Male	150 (54.3)	134 (89.3)	16 (10.7)	0.906**
Female	126 (45.7)	112 (88.9)	14 (11.1)	
Symptoms				
Dyspnea	145 (52.5)	126 (86.9)	19 (13.1)	0.210**
Cough	124 (44.9)	109 (87.9)	15 (12.1)	0.554**
Fever	107 (38.8)	98 (91.6)	9 (8.4)	0.296**
Weakness	72 (26.1)	63 (87.5)	9 (12.5)	0.605**
Myalgia	52 (18.8)	46 (88.5)	6 (11.5)	0.863**
Chest pain	34 (12.3)	32 (94.1)	2 (5.9)	0.554***
Diarrhea	26 (9.4)	23 (88.5)	3 (11.5)	1.000***
Loss of taste odor	17 (6.2)	15 (88.2)	2 (11.8)	1.000***
Throat ache	17 (6.2)	16 (94.1)	1 (5.9)	0.704***
Headache	12 (4.3)	12 (100)	0	0.374***
Loss of speech / movement	6 (2.2)	4 (66.7)	2 (33.3)	0.130***
Comorbidities				
Presence of any comorbidity	193 (69.9)	163 (84.5)	30 (10.9)	<0.001**
Hypertension	125 (45.3)	99 (79.2)	26 (20.8)	<0.001**
Coronary artery disease	83 (30.1)	58 (69.9)	25 (30.1)	<0.001**
Diabetes	74 (26.8)	50 (67.6)	24 (32.4)	<0.001**
Chronic lung disease	64 (23.2)	55 (85.9)	9 (14.1)	0.349**
Previous cerebrovascular disease	17 (6.2)	15 (88.2)	2 (11.8)	1000***
Chronic renal failure	12 (4.3)	8 (66.7)	4 (33.3)	0.030***
Malignancy	12 (4.3)	6 (50)	6 (50)	0.001***
Vital signs				
Fever (oC), median (IQR)	36.5 (36.12-37)	36.5 (36.1-36.9)	36.5 (36.3-37.32)	0.095*
Pulse (n/minute), mean (Std. deviation)	95.23 (20.49)	93.75 (20.2)	107.37 (19.11)	0.001****
Systolic blood pressure (mmHg), median (IQR)	140 (121.75-160)	140 (121.75-160)	127(107.5-150.25)	0.008*
Diastolic blood pressure (mmHg), median (IQR)	81 (70-94.75)	81.5 (70-95.25)	80 (67.75-90.25)	0.177*
Respiratory rate (n/minute), median (IQR)	15 (14-16)	15 (14-16)	16 (14-18)	0.002*
Oxygen saturation (%) median (IQR)	96 (94-98)	96 (94-98)	92.5 (87-97)	0.019*

*Mann-Whitney U test, **Pearson's chi-squared test, ***Fischer's exact test, ****Independent samples Student's t-test

and tiredness (n=72; 26.1%). One hundred ninety-three (69.9%) patients had a history of comorbid diseases. The most frequent comorbidities were hypertension (n=125; 45.3%), coronary artery disease (n=83; 30.1%), diabetes mellitus (n=74; 26.8%) and chronic lung disease (n=64; 23.2%) (Table 1). Thirty (10.9%) patients in the study lost their lives within 28 days. No statistically significant relation was observed between the 28-day all-cause mortality rate, and age, gender or symptoms at the time of ED visit. There was a statistically significant increase in the 28-day all-cause mortality rate in the presence of at least one comorbidity (p<0.001), hypertension (p<0.001), coronary artery disease (p<0.001), diabetes mellitus (p<0.001), chronic renal failure (p=0.03) and malignancy (p=0.001). The deceased patients had an elevated pulse and respiration rate, and reduced oxygen saturation compared to surviving patients (Table 1). The median of QTc interval on study patients’ initial ECGs was 390 (360-428) ms. The inspection of the rhythm on the ECGs of patient revealed that while sinus rhythm was prevalent (n=152; 55.1%), sinus tachycardia (n=75; 27.2%), atrial

fibrillation (n=25; 9.1%), supraventricular tachycardia (n=22; 8.0%) and ventricular tachycardia/fibrillation (n=2; 0.7%) were also present. Among the deceased patients, 40% had sinus tachycardia. Thirty-seven (13.4%) patients exhibited negative T waves, 9 (3.3%) had elevated/depressed ST segments and 8 (2.9%) had pathological Q waves. In the presence of QTc interval prolongation (p<0.001), ventricular tachycardia/fibrillation (p<0.001), left bundle branch block (p=0.001) and ST segment elevation/depression (p<0.001), 28-day all-cause mortality rate was significantly increased (Table 2). Involvement consistent with COVID-19 was observed on CCT scans of 173 (62.7%) patients. Distribution of pulmonary infiltrates was mostly peripheral (n=165; 59.8%) or bilateral (n=113; 40.9%). The most frequent findings on CCT scans of the patients were ground-glass opacities (n=142; 51.4%) (Figure 1), consolidation (n=51; 18.5%), pleural fluid (n=47; 17.0%) and multifocal nodules (n=22; 8.0%). The increase in 28-day all-cause mortality rate was statistically significant in the presence of severe (four or five lobes) (p<0.001), peripheral (p<0.001) and bilateral (p<0.001) lung involvement, ground-

Table 2. Patients’ electrocardiogram and chest CT findings

Total	28-day all-cause mortality			p-value
	All patients	Survivor	Deceased	
	n (%) 276 (100)	n (%) 246 (89.1)	n (%) 30 (10.9)	
Electrocardiogram				
QTc interval (milliseconds), median (IQR)	390 (360-428)	382.5 (360-420)	428 (358-630)	<0.001*
Sinus rhythm	251 (90.9)	226 (90)	25 (10)	0.167***
Right bundle branch block	14 (5.1)	13 (92.9)	1 (7.1)	1.000***
Left bundle branch block	18 (6.5)	11 (61.1)	7 (38.9)	0.001***
Atrial fibrillation	25 (9.1)	20 (80)	5 (20)	0.167***
Supraventricular tachycardia	22 (8)	19 (86.4)	3 (13.6)	0.718***
Ventricular fibrillation / tachycardia	2 (0.7)	0	2 (100)	<0.001***
T wave negativity	37 (13.4)	31 (83.4)	6 (16.2)	0.260***
ST segment elevation / depression	9 (3.3)	3 (33.3)	6 (66.7)	<0.001***
Presence of pathological Q	8 (2.9)	7 (87.5)	1 (12.5)	1.000***
Chest CT				
Pulmonary involvement	173 (62.7)	143 (82.7)	30 (17.3)	<0.001**
Peripheral distribution	165 (59.8)	136 (82.4)	29 (17.6)	<0.001**
Bilateral involvement	113 (40.9)	84 (74.3)	29 (25.7)	<0.001**
Ground-glass opacities	142 (51.4)	116 (81.7)	26 (18.3)	<0.001**
Consolidation	51 (18.5)	37 (72.5)	14 (27.5)	<0.001**
Pleural fluid	47 (17)	33 (70.2)	14 (29.8)	<0.001**
Multifocal nodule	22 (8)	17 (77.3)	5 (22.7)	0.075***
Air bronchogram sign	11 (4)	9 (81.8)	2 (18.2)	0.340***
Crazy-paving pattern	17 (6.2)	13 (76.5)	4 (23.5)	0.099***
Vascular enlargement	13 (4.7)	9 (69.2)	4 (30.8)	0.041***
*Mann-Whitney U test, **Pearson's chi-squared test, ***Fischer's exact test				

Table 3. Patients’ laboratory findings at admission and outcomes

Total	28-day all-cause mortality			p-value
	All patients n (%) 276 (100)	Survivor n (%) 246 (89.1)	Deceased n (%) 30 (10.9)	
AST (U/L), median (IQR)	23 (18-32)	23 (17-31.25)	30.5 (19.5-53.5)	0.022*
ALT (U/L), median (IQR)	16 (11.85-24)	16 (11.95-23.25)	18 (10.75-33.50)	0.490*
LDH (U/L), median (IQR)	264 (218-355.5)	260 (215-328.25)	359 (242.75-536.0)	0.003*
BUN (mg/dL), median (IQR)	16.8 (12.58-25.32)	16.40 (12.38-22.63)	26.29 (18.92-47.52)	<0.001*
Cr (mg/dL), median (IQR)	0.92 (0.73-1.17)	0.91 (0.73-1.10)	1.14 (0.81-2.27)	0.003*
Creatine kinase (U/L), median (IQR)	77 (53-115.75)	77 (54-111.25)	82.5 (36.25-185.75)	0.982*
D-dimer (µg/mL), median (IQR)	0.89 (0.46-1.93)	0.78 (0.40-1.62)	3.54 (1.36-3.54)	<0.001*
Troponin T (pg/mL), median (IQR)	12.35 (5.56-31.21)	11.57 (4.70-24.48)	53.21 (14.21-105.95)	<0.001*
CRP (mg/dL), median (IQR)	15 (3.14-15)	12.2 (3.14-45.67)	59.95 (29.07-109.25)	<0.001*
Ferritin (ng/mL), median (IQR)	112 (45-251.25)	106.60 (44.25-223.32)	143.8 (71.04-617.4)	0.020*
WBC (*10^9/L), median (IQR)	9.21 (6.84-12.44)	9.06 (6.71-11.82)	11.03 (7.87-18.02)	0.025*
Neutrophil count (*10^9/L), median (IQR)	6.46 (4.29-9.65)	6.27 (4.21-9.34)	9.32 (5.56-15.53)	0.009*
Lymphocyte count (*10^9/L), median (IQR)	1.63 (1.07-2.34)	1.71 (1.14-2.39)	1.05 (0.56-1.64)	<0.001*
Platelet count (*10^9/L), median (IQR)	234.5 (191.25-305.75)	236.50 (194-307.75)	219 (167.5-219)	0.270*
Hemoglobin (g/dL), mean (Std. deviation)	12.88 (2.15)	13.01 (1.99)	11.77 (2.98)	0.003***
MPV (fL), mean (Std. deviation)	9.89 (1.28)	9.86 (1.25)	10.18 (1.50)	0.033***
Outcome from ED				
Discharge	63 (22.8)	63 (100)	0	0.002**
Hospital ward admission	192 (69.6)	174 (90.6)	18 (9.4)	0.228**
Intensive care unit admission	19 (6.9)	9 (47.4)	10 (52.6)	<0.001**
Deceased in ED	2 (0.7)	0	2 (100)	
Discharged after hospitalization	183 (66.3)	183 (100)	0	
Deceased during hospitalization	28 (10.1)	0	28 (100)	
*Mann-Whitney U test, **Pearson’s chi-squared test, ***Independent samples Student’s t-test				

glass opacities ($p<0.001$), consolidation ($p<0.001$), pleural fluid ($p<0.001$), and vascular enlargement ($p<0.001$) on patient CCT scans (Table 2).

Compared to surviving individuals, deceased patients had higher levels of aspartate transaminase (AST) ($p=0.022$), lactate dehydrogenase (LDH) ($p=0.003$), blood urea nitrogen (BUN) ($p<0.001$), creatinine ($p=0.003$), D-dimer ($p<0.001$), troponin T ($p<0.001$), C-reactive protein (CRP) ($p<0.001$) and ferritin ($p=0.002$), white blood cell (WBC) count ($p=0.025$), neutrophil count ($p=0.009$), and mean platelet volume (MPV) ($p=0.033$), whereas lower lymphocyte count ($p<0.001$) and hemoglobin levels ($p=0.003$). The most pronounced increases were in the levels of D-dimer, CRP and troponin T. Deceased patients had approximately 4.5-fold higher D-dimer levels, and approximately 5-fold higher CRP and troponin T levels compared to those in surviving patients (Table 3).

An assessment of clinical outcomes following ED visit revealed that 192 (69.6%) of the study patients were admitted to a hospital ward, 19 (6.9%) were admitted to the intensive care unit, 63 (22.8%) were discharged and 2 (0.7%) lost their lives. Among the 211 hospitalized patients, 183 (66.3%) were discharged and 28 (10.1%) died during their hospital stay (Table 3).

Discussion

Determining disease severity, along with the COVID-19 diagnosis of patients visiting ED, is crucial for making the decision between discharge, admission to a ward and admission to the intensive care unit. Various studies have explored initial complaints, comorbidities, clinical findings, ECG findings, CCT imaging and laboratory results in order to distinguish high-risk COVID-19 patients. In one such study, advanced age, high number of affected lung lobes, elevated CRP levels at the time of the visit, chest pain/shortness of breath and a history of smoking were identified as independent risk factors for fatality [5]. Another study has found that age over 65, elevated cardiac troponin I levels, cerebrovascular disease and cardiovascular disease led to higher mortality rates. Among all the variables examined in the same study, a PaO₂ value of ≥ 80 mmHg was the only factor linked with survival [1]. Findings of another study have indicated a strong association between the mortality rate and the presence of hypoxemia necessitating oxygen support within three hours of presentation [6]. In a study involving COVID-19 patients in severe condition, male gender, SpO₂ $\leq 89\%$, respiratory rate of >30 /min and a diastolic pressure of ≤ 80 mmHg, were found to be related to significantly higher mortality rates [7]. In our study, 10.9% of the patients lost their lives within 28 days. There was no statistically significant difference between deceased and surviving patients in terms of age, gender or symptoms at the time of emergency department visit. Nonetheless, the 28-day all-cause mortality rate was significantly elevated in patients with at least one comorbid disease. Comparison of the initial vital signs of the study group noted at ED revealed that patients that died within 28 days had a higher pulse and respiratory rate, whereas oxygen saturation was lower than in surviving patients.

Patients may also develop cardiovascular complications associated with COVID-19. Fatal cardiovascular complications

seen in such patients include acute myocardial infarction, myocarditis, dysrhythmia, heart failure, cardiomyopathy, cardiogenic shock and venous thromboembolism [8]. Due to complications, ECG findings of these patients may include ST segment abnormalities, QT prolongation, conduction abnormalities and ventricular arrhythmias. Accordingly, patients that exhibit cardiac symptoms and ECG abnormalities should be carefully examined with regards to COVID-19 related cardiac complications [9]. Patients with severe COVID-19 had a higher risk of arrhythmia complications [10]. One study has shown that patients had higher mortality rates if their ECG findings included one or more atrial premature contractions, right bundle branch block or intraventricular block, ischemic T-wave inversion, and nonspecific repolarization. ST elevation has been found to be rare at the time of the initial hospital visit [6]. Another study has reported the presence of sinus tachycardia and ventricular arrhythmia on the ECG as independent risk factors for fatality [11]. Among our study group, 40% of the deceased patients had sinus tachycardia. The 28-day all-cause mortality rate was significantly elevated in the presence of QTc interval prolongation, ventricular tachycardia/fibrillation, left bundle branch block and ST segment elevation/depression.

rRT-PCR testing has become a standard tool for the diagnosis of COVID-19. However, no difference has been observed in viral load between symptomatic and asymptomatic patients. The effectiveness of the rRT-PCR test for COVID-19 diagnosis at ED is limited due to its low positivity rate and long procedure time [12,13]. CCT imaging is a favorable diagnostic tool, as it is readily accessible, and yields fast and robust results. The typical radiological features of COVID-19 include bilateral, multifocal, and multilobar ground-glass opacification with patchy consolidation, a peripheral/subpleural or posterior distribution (or both), mainly in the lower lobes. The combination of CCT and repeated Reverse Transcription-Polymerase Chain Reaction (RT-PCR) testing may be beneficial for the diagnosis of COVID-19 in the setting of strong clinical suspicion [14]. In a cohort of 1,014 patients with a 71% rRT-PCR positivity rate, 98% of the patients that tested positive exhibited CCT findings indicative of COVID-19 lung involvement (bilateral ground-glass opacities and consolidations distributed peripherally especially in lower lobes) [15]. Another study has shown that 86% of the patients with positive rRT-PCR results had bilateral ground-glass opacities on their CCT scans [16]. While early-stage patients' CCT scans show small plaque shadows and interstitial changes, severe cases display multiple ground-glass opacities, infiltration and consolidation [17]. Another study has reported that a higher number of involved lobes (5 involved lobes) within the first week of disease onset were associated with elevated mortality rates [18]. In spite of its high sensitivity, CCT imaging may not be always useful for diagnosis or exclusion of COVID-19. Especially in asymptomatic patients, in those with mild symptoms and in those without pneumonia, CCT is not an effective diagnostic tool. Further disadvantages of CCT include high radiation emission, unavailability in smaller hospitals and requirement for radiologist interpretation [12,13,17]. In our study, CCT scans of 62.7% of the patients exhibited lung involvement concordant with COVID-19. The most common CCT findings were ground-glass opacities, consolidation, pleural

fluid and multifocal nodules. Severe (4 or 5 lobes), peripheral and bilateral involvement of lobes, ground-glass opacities, consolidation, and vascular enlargement on CCT scans were associated with rise in 28-day all-cause mortality rates.

Laboratory tests are nonspecific for the diagnosis of COVID-19. The most frequent laboratory findings in COVID-19 patients are normal/low lymphocyte count, coagulopathy, and increased levels of CRP, erythrocyte sedimentation rate, LDH, aminotransferase and ferritin. Conversely, procalcitonin levels are typically normal. Elevated levels of D-dimer, troponin I, ferritin, LDH and IL-6, as well as low basal lymphocyte count have been found to be related to worse prognosis [12,13,18-20]. According to another study, a four-fold increase in D-dimer levels compared to the time of admission could be used as an effective predictor of in-hospital fatality [21]. In agreement with the literature, our study showed that deceased patients had higher levels of AST, LDH, BUN, creatinine, D-dimer, troponin T, CRP, ferritin, WBC, neutrophil and MPV, and lower levels of lymphocyte and hemoglobin than those in surviving patients. The most significant increases were in the levels of D-dimer, CRP and troponin T. Compared to survivors, the deceased patients had 4.5-fold higher D-dimer levels, as well as 5-fold higher CRP and troponin T levels.

In conclusion, diagnosing and determining the severity of COVID-19 at ED can be achieved by evaluating vital signs, comorbidities, and findings of ECG, laboratory tests and CCT. At the time of ED visit, the presence of at least one comorbidity, high pulse and respiratory rate, reduced oxygen saturation, ECG findings of sinus tachycardia, QTc interval prolongation, ventricular tachycardia/fibrillation, left bundle branch block and ST segment elevation/depression, CCT findings of severe lung involvement (four or five lobes), peripheral/bilateral lobe involvement, ground-glass opacities, consolidation, pleural fluid and vascular enlargement, elevated levels of D-dimer, CRP and troponin T were associated with higher 28-day all-cause mortality rates.

Scientific Responsibility Statement

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

Animal and human rights statement

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. No animal or human studies were carried out by the authors for this article.

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Conflict of interest

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